Myocardial Contusion in An Eight-year-old Child

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THE MAJORITY OF nonpenetrating crushing injuries of the heart occur in adults, for they are more likely to be involved in traffic and industrial accidents than are children.[3,6,9] The forces that cause such injuries include: Sudden deceleration of the body against a blunt unyielding object such as a steering wheel, compression by two converging vehicles, impact of a high pressure jet or blast that throws the body against an unyielding surface,[3] and the impact of a blunt flying object such as a baseball or a fist. Nonpenetrating chest trauma leading to myocardial injury has been reported more rarely in the pediatric age group.[7,8]

It is the purpose of this report to present a case of blunt injury to the heart as a result of an accident in a fairly common play situation involving grade school children, and to bring attention to various aspects of diagnosis and management.

Report of a Case

An 8-year-old boy was struck between the 5th and 6th ribs by the tip of a plastic toy "BAT-BAT"* thrown by his 13-year-old brother at close range. The boy screamed, collapsed to the ground and for one or two minutes appeared unconscious. He became ashen and diaphoretic and, after regaining consciousness, began thrashing about with intense anterior chest pain. He was immediately taken to a local medical clinic where his heart rate was found to be 50 per minute and the systolic blood pressure was 70 mm of mercury. He responded promptly to emergency treatment with intramuscular epinephrine and oxygen inhalation.

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*Manufactured by Reuben Klamer, Beverly Hills, California.

Because of clinical and electrocardiographic evidence of cardiac trauma, he was referred to the UCLA Medical Center for close observation about 28 hours after the injury.

On examination he was observed to be a healthy, alert 8-year-old boy in no distress. There was a small tender erythematous area in the 5th intercostal space 3 cm left of the sternal margin corresponding to the area of impact of the missile. The blood pressure was 90/65 mm of mercury in the right arm, the heart rate 70, respirations 16 per minute and temperature 37.6°C orally. The heart tones were normal except for a short, low grade, apparently innocent, ejection murmur at the pulmonic area. The peripheral pulses were of good quality.

Roentgenographic examination did not reveal any bony or soft tissue abnormalities. The cardiac silhouette was at the upper limit of normal with a cardio-thoracic ratio of 0.53.

The patient remained in the hospital for seven days of strict bed rest. Electrocardiographic monitoring and night-time use of an oxygen tent were carried out on the first two days as precautionary measures. At no time were there signs of cardiac decompensation or angina pectoris, nor was there any abnormality to auscultation. The patient was then released with advice of limited physical activity at home. On visits to the hospital 14 days and 28 days after the incident, the patient remained asymptomatic as he gradually increased his physical activity to a normal level.

Electrocardiographic Findings

Figure 1 shows serial electrocardiograms in chronological order. Shortly after the accident there were sinus bradycardia, right bundle branch block (RBBB) with QRS interval of 0.14 sec, ST segment elevation of the "ischemic type" in V_2 through V_4 with associated deep inversion of T waves, and T waves in V_5 and V_6 were tall and peaked. Lead aVL showed a low voltage RS pattern with a slowly rising ST-T.

Approximately 18 hours after the incident, the heart rate had increased to 80 per minute. The QRS interval had narrowed to 0.06 second and the amplitude of R waves was definitely increased in leads II, III and aVF. There was no evidence of RBBB. ST segment elevation persisted with less inversion of T waves in V_2 through V_4. There was ST elevation of a junctional type in leads I, V_5 and V_6. Ectopic ventricular beats presumably of left

ventricular origin were occasionally noted. aVL at this time revealed a QR pattern with Q wave duration of 0.04 second.

Subsequent tracings showed further increase in amplitudes of R waves in Leads II, III, aVF and V_5 and V_6 and corresponding increase in S wave amplitudes in Leads V_1 through V_4. ST-T changes in V_2 and V_4, described previously, tended to improve gradually throughout the hospital stay. Ectopic beats were not seen after the day following injury.

Serum Enzyme Studies

Slight to moderate elevations of serum glutamic oxaloacetic transaminase (SGOT) and lactic dehydrogenase (LDH) levels were noted the day after injury. The former returned to normal by the second day and the latter by the sixth day.

TABLE 1.—*Serial Serum Enzyme Levels After Myocardial Contusion in the Case Presented*

Time Lapse After Injury	*28 Hours*	*3 Days*	*4 Days*	*5 Days*	*6 Days*	*7 Days*
Serum glutamatic oxaloacetic transaminase	48	24	....	24	18	15
Lactic dehydrogenase	487	375	365	355	336	325

Normal range for SGOT in our institution: 10 to 40 units.
Normal range for LDH: 100 to 350.

The results of enzyme studies are summarized in Table 1.

Comment

Blunt trauma can damage different parts of the heart. Hemopericardium and fibrinous pericarditis can be detected on the basis of clinical evidences of cardiac tamponade or pericardial friction rubs combined with ancillary diagnostic aids. Nonpenetrating trauma has been known to produce avulsion of chordae tendinae, of valve leaflets or of papillary muscles, resulting in sudden onset of valvular incompetence.[8,9] Ventricular septal defects of traumatic origin have been reported in association with both penetrating and non-penetrating injuries.[4] Ventricular pseudoaneurysm[5] and rupture of cardiac chambers[9] following chest trauma have been described.

The present case belongs in a larger group of cardiac trauma—myocardial contusion with or without concomitant ischemic necrosis. Coronary artery damage might be implicated, either by actual severance of the vessel or extensive perivascular hematoma compressing it. Parmley and associates[9] reported in 1958 that actual intraluminal coronary thrombosis secondary to trauma was very unlikely without antecedent atherosclerosis.

Although electrocardiographic findings in the present case were suggestive of localized anterior

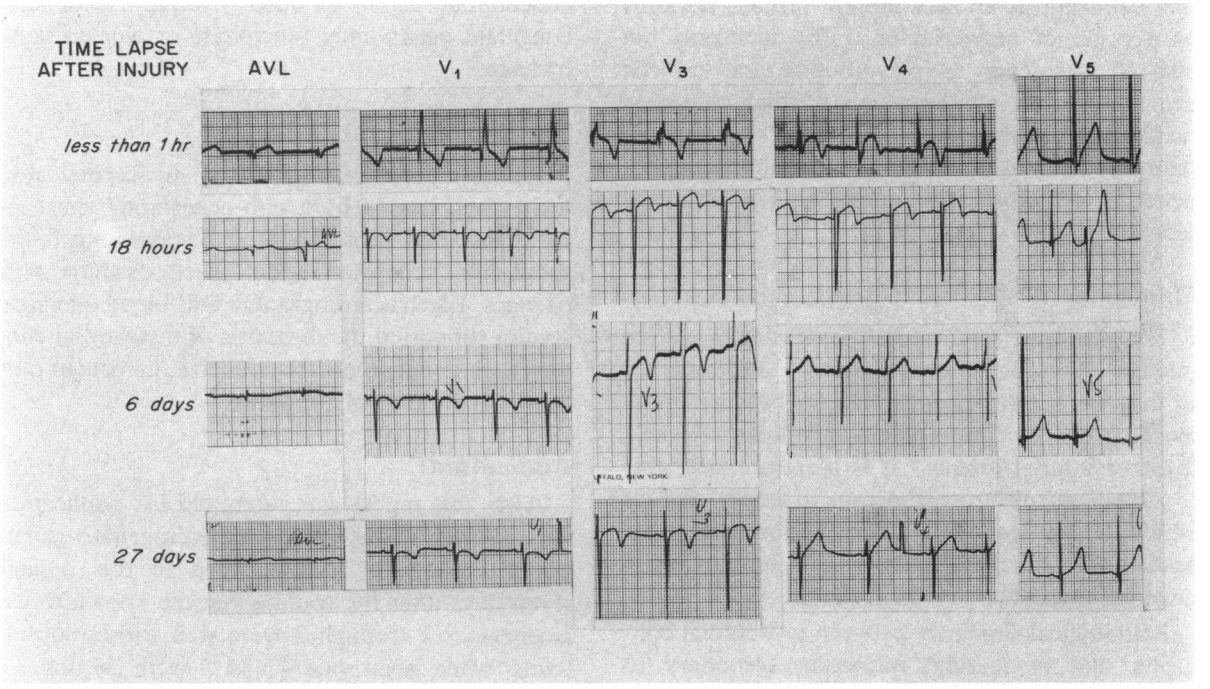

Figure 1.—Serial electrocardiograms following blunt trauma to the anterior chest.

wall ischemic necrosis, it is impossible to differentiate tissue damage due entirely to mechanical trauma from infarction secondary to damage of the left coronary artery branch.

Sinus bradycardia, complete or incomplete bundle branch block and ST segment elevation have been described in myocardial contusion by Barber[2] and by Goldring and associates.[7] Transient high amplitude or peaked T waves have also been observed in 10 to 15 per cent of traumatic myocardiopathy according to these investigators. Low voltage QRS complexes (seen in six of eighteen patients) were regarded as strong evidence of myocardial injury.

Though reports of long term follow-up of myocardial contusion are few, most observers agree that prognosis is usually good in mild to moderate cases. Akenside[1] in 1764 described a boy struck by a plate who died unexpectedly six months later, presumably from acute arrhythmia arising from localized full-thickness necrosis of the left ventricle. Bregani[3] reported a young man struck and thrown by a high pressure jet who had recurrent angina pectoris and electrocardiographic evidence of myocardial ischemia for three months following the original injury. In another case, residual calcification presumably of a mural thrombus was discovered many years after the original accident.

Finally, one should bear in mind that myocardial contusion is an ever-present menace not only as a result of industrial or traffic accidents, but also in accidents in playgrounds and athletic events. A case of severe myocardial contusion resulting from the act of "spearing" (use of the helmeted head as a battering ram against an opponent) in a football scrimmage has been reported recently.[10]

Management

Diagnosis of myocardial contusion may be missed either because symptoms[10] are transient as they were in the present case, or because of the masking effect of more overt symptoms referable to other injured organs.[9] It is therefore advisable to obtain an electrocardiogram in every case of violent chest trauma. If any abnormality suggestive of myocardial injury is seen, serial tracings must be obtained.

Pathological similarity between myocardial contusion and myocardial infarction secondary to coronary artery disease leads us to regard complete bed rest as a "must" for a period of time sufficient to allow for cessation of necrosis of injured tissue and establishment of healing process. This is ten days to two weeks with mild damage, as in the present case, and up to six to eight weeks in severely injured patients. Severity of associated injuries may also dictate the duration of bed rest. Electrocardiograms, serum enzyme studies including transaminase, lactic dehydrogenase and creatine phosphokinase[11] and erythrocyte sedimentation rates obtained at appropriate intervals serve as useful guides. Oxygen therapy may be a useful adjunct in reducing cardiac work in the initial period. Continuous monitoring of the electrocardiogram is important in early detection and treatment of life-threatening arrhythmias. Frequent assessment of pulse, respiration rate and blood pressure would not only alert one to congestive heart failure at its incipient stage but might lead to earlier detection of sudden rupture of ventricular septum or a papillary muscle. These complications may occur hours or days after the initial injury[8] and are known to cause acute congestive heart failure or pulmonary hypertension or both. Such lesions often require operative repair with extracorporeal circulation.

Anticoagulation is probably not indicated because of the very rare incidence of coronary thrombosis secondary to trauma, especially in young patients. Furthermore, in the presence of concomitant injury to other viscera, use of anticoagulant agents may precipitate or worsen hemorrhage.

Summary

An 8-year-old boy in whom myocardial contusion was caused by a non-penetrating chest injury recovered promptly after an alarming initial shock-like state and serial electrocardiographic changes. Electrocardiographic and blood chemical studies supported the diagnosis of myocardial contusion or ischemic necrosis despite the benign outward appearance of the lesion.

Addendum:

After this report was submitted for publication serum levels of lactic acid dehydrogenase isoenzymes 1 through 5 determined in this patient seven days after the trauma became known to us. Isoenzymes 1 through 3 were well within normal limits, while isoenzyme 4 and 5 were 26 and 37 units (normal range for both: 0 to 15 units). Ele-

vation of LDH isoenzyme 5 is highly specific for myocardial injury.[12]

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Seminal Vesicular Cyst

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THE PURPOSE of this paper is to report the diagnosis and successful treatment of a seminal vesicular cyst in a patient and to review the literature which establishes this as the eighth such case reported.

Approximately 20 cases of seminal vesicular cysts have been reported. In many of the older reports the lesions apparently were Mullerian duct cysts.[2,5,6,18,20] More recent reports were based on roentgenographic evidence only, or on surgical demonstration of a cyst "in the area of the seminal vesicle."[7,8,12,13] Careful review of the literature disclosed only seven cases of clinically discovered and anatomically proved cysts of the seminal vesicle.*

Englisch[4] believed that seminal vesicular cysts developed from inflammatory closure of small diverticula. He also described various other types of cysts found in this area, such as those arising from Wolffian duct remnants and generally located in the region of the vas deferens at the posterolateral aspect of the bladder, as noted in the report published by Lund and Cummings.[16] Other cysts arising from Mullerian duct remnants are usually midline in position and are attached to the posterior bladder wall; these are further discussed by Coppridge.[1] Cystic dilatation of the utricle may be secondary to stricture of its orifice as was noted in a case reported by Lubash.[15]

Voelcker,[22] Schwarzwald,[19] and Lloyd and Pranke[14] cautioned against making the diagnosis of seminal vesicular cyst solely from clinical findings and roentgenographic studies. They stressed that anatomic confirmation should be accomplished by surgical operation, demonstrating the cyst to be an integral part of the seminal vesicle. Spermatozoa are usually found in the cystic fluid, but their presence is not diagnostically imperative. Subsequent histopathologic confirmation of seminal vesicular tissue in the wall of the cyst represents final verification.

Report of a Case

A 36-year-old white man, an avowed homosexual, entered Mount Zion Hospital and Medical Center, San Francisco, 24 August 1966 with a six-week history of persistent, painless bloody urethral discharge which made "nickel-sized bright red spots" on his underwear. For several days before admission he had had vague suprapubic discomfort. There were no lower urinary tract symptoms nor gross hematuria nor history of tuberculosis.

The patient had been put in hospital by another physician five weeks previously for evaluation of the same complaints. Microscopic hematuria was present then. Endoscopy had revealed an anterior urethral stricture which was promptly dilated. Retrograde pyelography at that time demonstrated bilateral renal calculi. Our urologic consultation was sought by the patient because the urethral discharge persisted. Past genitourinary history in-

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*Reference Nos. 3, 9, 10, 14, 21, 23.